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ORIGINAL PAPER

REHABILITATION AFTER OPERATIVE RECONSTRUCTION OF SHOULDER FOR RECURRENT FORWARD DISLOCATION

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From the Royal Air Force Medical Rehabilitation Units, Chessington and Collaton Cross

"RECURRENT dislocation of the shoulder can only be dealt with satisfactorily by operation" (Wiles, 1951a).

Many operative procedures have been described for the treatment of recurrent dislocation of the shoulder, the techniques most frequently performed being the Bankart and the Putti–Platt. It is claimed that the essential basis for successful operation lies in buttressing the anterior aspect of the joint and preventing the head of the humerus riding out of the glenoid cavity at the extreme of external rotation. Defects of the head of the humerus and of the glenoid labrum allow the head to dislocate before full external rotation is reached, and thus successful repair involves some restriction of external rotation (Watson-Jones, 1955a). The principle of the Bankart operation is the repair of the defect in the glenoid labrum. In the Putti–Platt operation the subscapularis is "double-breasted", thus tightening the structures in front of the joint. Some surgeons, however, believe that the main benefit of repair operations is the fibrosis resulting in the front of the capsule from the surgical trauma.

After operation the limb is usually bound to the trunk with adhesive bandage so that the shoulder is adducted and internally rotated. This protects the tightened capsule from external rotation strain during healing. The period of immobilization varies from four to six weeks, depending on the preference of the surgeon and the age of the patient, and is followed by active exercises.

It is generally accepted (*Lancet*, 1956) that operative repair of recurrent dislocation necessarily results in some permanent limitation of shoulder movement. Wiles (1951b) expects limitation of both abduction and external rotation, whereas Watson-Jones (1955b) records full abduction after three to four months, but permanent restriction of external rotation to about 40 degrees. In order to avoid the permanent restriction various techniques have been evolved, in particular that of stapling the damaged portion of the glenoid labrum.

The value of planned rehabilitation following operation for recurrent dislocation of the shoulder was stressed by Rudd and Haydock (1952) in reference to Bankart operations. Patients receiving planned rehabilitation achieved full power and movement and returned to duty in ten weeks after operation, whilst those not receiving supervised exercises took five months to become fit for duty.

PRESENT SERIES

In the Services recurrent dislocation of the shoulder is usually treated by a Putti–Platt type of operation, with repair of the defect in the glenoid labrum when present and technically feasible. After operation patients are transferred to rehabilitation units.

Between 1953 and 1956 a total of 100 Service patients (mostly between 19 and 30 years old) were transferred to the Royal Air Force Medical Rehabilitation Units at Chessington and Collaton Cross following this operation. The commonest cause of the original dislocation was a fall, and among sports Rugby football was the most frequent cause.

Recorded Cause of Original Dislocation

	No. of Patients		No. of Patients
Fall	17	Swimming	2
Rugby football	11	Road accident (other than motor-cycle)	2
Soccer	8	Direct violence	4
Motor-cycle accident	5	Billiards	1
Boxing	5		
Gymnastics	4		

Number of Dislocations and Length of History

The number of times the dislocation occurred ranged from 2 to 60 (average 6), with an average length of history of 22 months.

	Number of Dislocations Before Operation			
	Less than 3	3–5	6–10	More than 10
No. of patients	5	39	27	29

	Length of History (years)			
	Less than 1	1–2	2–3	More than 3
No. of patients	11	56	16	17

Duration of Disability and Period of Immobilization

The average total period of disability from operation to return to duty in this series was 12 weeks. The period of immobilization ranged from 3 to 8 weeks; in the majority it was 6 weeks. The average period of immobilization was 5½ weeks, and the average time spent at the rehabilitation unit 6 weeks. Patients were transferred to the rehabilitation unit between the third and eighth weeks after operation.

	Time Immobilized (weeks)					
	3	4	5	6	7	8
No. of cases	23	13	15	43	3	3
Average time at M.R.U. (weeks) ..	6½	6½	6	7	5	3

Mobilization Time

The "mobilization time" is reckoned from removal of strapping immobilization until the patient is returned to duty, whether the time is spent at the rehabilitation unit or not.

	Mobilization Time (weeks)								
	Less than 4	4	5	6	7	8	9	10	More than 10
No. of cases	8	18	15	13	14	9	6	6	11

Neither the length of the history nor the number of times the dislocation recurred significantly influenced the mobilization time, but prolonged immobilization lengthened the time required for rehabilitation.

EFFECT OF PERIOD OF IMMOBILIZATION ON REHABILITATION

Immobilization Period	No. of Cases	Average Time before Transfer to M.R.U. (weeks)	Average Time at M.R.U. (weeks)	Average Mobilization Time (weeks)	Average Total Disability Time (weeks)
6 weeks or more ..	49	6	7	7	13
4–6 weeks	36	5	6	7	11
Less than 4 weeks ..	15	4	5	6	9

REHABILITATION PROGRAMME

Patients transferred to the rehabilitation unit early are taught "static deltoid" contractions while the arm is still immobilized; movements of the wrist and fingers are encouraged by occupational therapy and exercises. Four weeks after operation the strapping is removed but the arm is left in a sling. Active movements of the shoulder are encouraged under individual supervision. During the fifth and sixth weeks after operation the arm is kept in a sling except while undergoing active treatment. Occupational therapy encourages active shoulder movements, but the sling reminds the patient to restrict external rotation. By the end of the sixth week the patient will usually have regained full elbow movement and have at least 60 degrees of active shoulder abduction. The sling is now discarded and the patient starts full class exercise routines.

Patients who are transferred at six weeks after operation are ready to start class work immediately. This is based on free movements of the shoulder in the painless range, the range steadily increasing through the period of rehabilitation. No movement is forced or assisted, and external rotation is never allowed.

Fifteen patients were immobilized by means of a sling only, and transferred to the rehabilitation unit in the third or fourth week after operation. These patients regained movements very quickly, usually including about 50 degrees range of external rotation, although a full rehabilitation programme was not introduced until the fifth or sixth week after operation. (It was in this group that laxity of the shoulder and lack of confidence tended to occur.)

Patients are exercised stripped to the waist, so that incorrect and trick movements, particularly elevation of the shoulder simulating abduction, are detected and avoided.

The early class exercises for the shoulder are "loose arm swings" with the arms hanging freely at the sides or slightly forwards with the trunk flexed. These exercises form a small part of the class routine, which aims also at establishing full elbow, wrist, and hand movements as well.

Progression to abduction, forward flexion, and extension introduces the effect of gravity as the patient regains control of his shoulder. Throughout rehabilitation of shoulder disabilities the mobilizing exercises are restricted to the free, painless range; no forced, assisted, or passive exercises are allowed. The free, painless range steadily increases as the effect of gravity increases. The duration of specific shoulder exercises is increased as recovery proceeds, and thus power, mobility, and endurance are increased *pari passu.*

Games (such as quoits and darts) which entail abduction and rotation from full internal rotation to the neutral position are of particular value, but heavy games and ball games are avoided during the first four weeks of rehabilitation. Archery and swimming with "dog-paddle" or breast stroke are introduced during the ninth and tenth weeks after operation. If patients require a particularly powerful shoulder for their job, or if the power is poor (which is rare), then weight-lifting is introduced.

This regimen avoids stretching of the structures in front of the joint and allows external rotation, if it is going to return, to return on its own.

Patients transferred before immobilization was completed required, on an average, two weeks less mobilization than those transferred after removal of immobilization.

EFFECT OF TIME OF TRANSFER TO REHABILITATION UNIT ON MOBILIZATION TIME

	No. of Patients	Average Mobilization Time (weeks)
Patients transferred to rehabilitation unit after completion of immobilization period	39	8
Patients transferred to rehabilitation unit before completion of immobilization	61	6
Total	100	6½

That this regimen is effective is shown by the results recorded at the end of rehabilitation in 153 patients treated at Chessington and Collaton Cross since 1951 (including 53 quoted by Wynn Parry, 1954).

	Percentage of Patients
Returned to full service duty	100
With full internal rotation	100
With full forward flexion	95
With full abduction	88
With external rotation of 30 degrees or more	60

AFTER-CARE

When patients leave the rehabilitation unit they are advised to avoid violent games and similar activities (particularly Rugby football and high-jumping) for a further three months. Patients are also advised of the need to retain the power of the shoulder muscles by continuing the exercises performed at the rehabilitation unit. They must also train properly before resuming sport.

COMPLICATIONS

In this series post-operative complications were very few—one case each of post-operative atelectasis, wound sepsis, and peripheral nerve lesion.

A small number of patients are slow to regain movement and power. Active movements assisted by a physiotherapist are introduced at the eighth week after operation when it is clear that progress is slower than it should be. The cause may be either apprehension on the patient's part or a particularly "tight" repair. A few cases of laxity of the shoulder have been associated with the introduction of active movements in the first few weeks after operation. These patients have regained movements with unusual rapidity, but failed to regain power, and complain of a sensation of instability and lack of confidence. The important complication of this disability is a further recurrence of the dislocation.

Three patients in this series had had a previous operation, this being the Putti–Platt in two cases and the Bankart in one case.

The ultimate assessment of results can be made only on long-term follow-up, unfortunately not possible in this series.

COMMENT

The object of operative reconstruction of the shoulder is to prevent further dislocations and to restore the patient's confidence in his shoulder. The prevention of recurrent dislocations can be assessed only by detailed analysis of long-term follow-up records, but the short-term result can be based on the patient's subjective assessment of his shoulder. The three main operative techniques are the Bankart repair of glenoid defects, stapling of glenoid defects, and the buttressing type of operation (Putti–Platt).

Lack of confidence with a feeling of instability occurs only after Bankart operations or after too early mobilization following a Putti–Platt repair. The slight restriction of movement associated with "buttressing" was a psychological advantage. It is undoubtedly true that the period off duty is shorter with the Bankart type of repair. A recent review by DuToit and Roux (1956) of the results of stapling glenoid labrum defects gave the average disability period as

six weeks. More recently the tendency has been to reduce this further, but the time required for shoulder power to build up is not given. Full recovery was recorded in 68% and "slight limitation" in 23% at follow-up; 66% of the patients were known to have resumed sport.

In our experience, however, buttressing operations give consistently good, stable results with full confidence. With full-time planned rehabilitation there is no need for any functional restriction to remain.

SUMMARY

Full-time rehabilitation can give functional results after "buttressing" operations on the shoulder which cannot be obtained by the usual practice of out-patient physiotherapy. The regimen starts with "static exercises" for the deltoid while the patient is still immobilized with adhesive bandages in adduction and internal rotation. The strapping is removed between four and six weeks after operation and the shoulder and elbow are exercised freely under close individual supervision, a sling being worn at all other times.

Class exercises are introduced six weeks after the operation to mobilize the shoulder, but avoiding external rotation.

With this regimen the patient should be fit to return to full Service duty 10–12 weeks after operation with full abduction, forward flexion, internal rotation, and 30 degrees or more of external rotation, and with full confidence in his shoulder.

ACKNOWLEDGMENTS

It is a pleasure to acknowledge the interest and co-operation of our orthopaedic colleagues in all three Services. We would like to thank, in particular, Air Commodore L. M. Crooks, Wing Commander D. M. Keir, Colonel A. McMillan, Lieut.-Colonel P. R. Wheatley, Commander W. S. Wilson, and Lieut.-Commander T. C. Law.

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ORIGINAL PAPER

THE EFFECT OF CORTICOSTEROIDS ON THE SKIN RESPONSE TO ULTRAVIOLET LIGHT IN RHEUMATOID ARTHRITIS

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COUNTER-IRRITATION by ultraviolet light (U.V.L.) is a physical method commonly used in the local treatment of joints affected by the rheumatic diseases. In the case of rheumatoid arthritis corticosteroid drugs are now frequently prescribed, and it seems likely that the skin response to U.V.L. might well be modified by these drugs, in view of their known anti-inflammatory effect. Lovell *et al.* (1953) were unable to demonstrate any significant alteration in the immediate skin reaction to injury while the subject was taking cortisone, but both Jarvinen (1951) and Holti (1956) consider that cortisone greatly reduces the intensity and duration of the skin reaction to U.V.L.

Theoretically, it is possible that a skin test for U.V.L. sensitivity might be given to a patient whose skin response has been modified by steroids. If the drug should be withdrawn whilst counter-irritation was continued there would be a danger of producing a serious burn, since repeated test doses of U.V.L. are unlikely to have been given.

Very few subjects with rheumatoid arthritis have been considered in previous experiments, and the purpose of the present work was to investigate the modifying effect (if any) of cortisone and other corticosteroids on the skin response of U.V.L. in cases of rheumatoid arthritis.

METHOD

Ten in-patients (8 female, 2 male) with an age range of 40–60 years were given corticosteroids to control active rheumatoid arthritis. None was receiving gold or phenylbutazone, although all were taking aspirin preparations. None had skin lesions. Repeated tests for the presence of L.E. cells were carried out in doubtful cases, all with negative results. The E.S.R. (Westergren) and haemoglobin concentration were measured weekly, and in eight of the ten subjects a satisfactory fall in E.S.R. and rise in haemoglobin level were found during the steroid administration.

All patients were initially given moderately high daily doses of steroids, in divided doses, by mouth, and these were gradually tapered off to reach a main-

tenance level in four to six weeks. One subject was given cortisone, three prednisone, three prednisolone, and three hydrocortisone.

Before initiating steroid therapy two preliminary skin tests (*a* and *b* in Table II) with U.V.L. were carried out at four-day intervals. The steroid course was started and four to seven days later the U.V.L. skin tests were repeated. They were repeated at approximately weekly intervals on two to three occasions. Two subjects were tested only once after steroid administration had begun.

The source of U.V.L. in every case was an air-cooled mercury vapour lamp at a distance of 18 inches from the skin, and both the lamp and reflector were cleaned with spirit before each exposure. Stabilization of U.V.L. output was achieved by switching on the unit ten minutes before use. Careful adjustment was made to ensure that the incident light fell at right-angles to the surface exposed.

In every case the site used was the back between the shoulder and the upper lumbar region. The skin was first cleaned with soap and water and then with surgical spirit, and lightly dried.

A thick black paper shield, with six square holes 1·5 cm. square, and each separated from the next by a distance of 2·5 cm., was used to expose the test areas of skin successively to 7, 15, 30, 45, 60, and 90 seconds of U.V.L. The rest of the body was adequately protected.

The same team of two observers (one doctor and one physiotherapist) noted the erythemal reactions in all cases at 6, 12, 24, and 72 hours. The erythemal reaction was graded according to the criteria given in Table I.

TABLE I

CRITERIA FOR GRADING SKIN REACTION

Erythema Grade	Latent Period (Hours)	Degree of Reaction	Duration of Erythema	Pigmentation	Peeling
E1	12–24	Faint pink	12 hours	—	—
E2	8–10	Pink	24 hours	Faint	Powdery
E3	6–8	Red	5–7 days	Deep	Exfoliation
E4	4–6	Fiery red	7 days	Deep	Blister

RESULTS

The results, given in Table II, show that the skin response to ultraviolet light is unmodified by the systemic administration of steroid preparations. None of the ten subjects showed any evidence that the steroid reduced the inflammatory response to any significant extent.

In eight subjects the degree of erythema produced by specified test doses of U.V.L. in each individual was substantially unchanged throughout the experiment. In the other two cases (Subjects 2 and 5) sensitivity was apparently increased while steroids were being administered.

TABLE II

ERYTHEMA REACTION OF SKIN TO U.V.L. BEFORE AND DURING STEROID THERAPY

Subject	Time of Test (Weeks)	Exposure Time (Seconds)						Steroid Given and Daily Dosage (mg.)
		7	15	30	45	60	90	
1. Male aged 48	Preliminary *a*	E1	E1	E1	E2	E2	E2	0
	Preliminary *b*	E1	E1	E2	E2	E2	E2	0
								Cortisone
	<1	E1	E1	E1	E2	E2	E2	70·0
	1–2	E1	E1	E2	E1	E2	E2	50·0
	2–3	E1	E1	E1	E1	E1	E1	37·5
	3–4	E1	E1	E2	E2	E2	E2	12·5
	>4	E1	E2	E2	E2	E2	E2	10·0
2. Male aged 61	Preliminary *a*	E1	E2	E2	E2	E2	E2	0
	Preliminary *b*	E1	E2	E2	E2	E2	E2	0
								Prednisolone
	<1	E1	E2	E2	E2	E3	E3	30·0
	1–2	E1	E2	E3	E3	E3	E3	20·0
	2–3	E1	E1	E1	E1	E1	E1	15·0
	3–4	E1	E1	E2	E3	E3	E3	12·5
	>4	E1	E2	E3	E3	E3	E3	10·0
3. Male aged 57	Preliminary *a*	E1	E2	E2	E2	E3	E3	0
	Preliminary *b*	E1	E1	E2	E3	E3	E3	0
								Hydrocortisone
	<1	E1	E1	E1	E1	E1	E1	120·0
	1–2	E1	E2	E3	E3	E3	E3	100·0
	2–3	E1	E1	E1	E1	E1	E1	80·0
	3–4	E1	E2	E3	E3	E3	E3	60·0
	>4	E1	E2	E3	E3	E3	E3	50·0
4. Male aged 57	Preliminary *a*	E1	E1	E2	E3	E3	E3	0
	Preliminary *b*	E1	E1	E2	E3	E3	E3	0
								Hydrocortisone
	<1	E1	E1	E1	E1	E1	E1	120·0
	1–2	E1	E1	E2	E2	E3	E3	100·0
	2–3	E1	E1	E1	E1	E1	E1	80·0
	3–4	E1	E2	E3	E3	E3	E3	60·0
	>4	E1	E1	E2	E3	E3	E3	50·0

TABLE II—*continued*

Subject	Time of Test (Weeks)	Exposure Time (Seconds)						Steroid Given and Daily Dosage (mg.)
		7	15	30	45	60	90	
5. Male aged 57	Preliminary *a*	E1	E2	E2	E2	E3	E3	0
	Preliminary *b*	E1	E1	E2	E2	E3	E3	0
								Prednisolone
	<1	E1	E1	E1	E1	E1	E1	30·0
	1–2	E1	E2	E3	E3	E3	E3	25·0
	2–3	E1	E1	E1	E1	E1	E1	20·0
	3–4	E1	E2	E3	E3	E3	E3	15·0
	>4	E1	E2	E3	E3	E3	E3	15·0
6. Male aged 51	Preliminary *a*	E1	E1	E2	E2	E2	E2	0
	Preliminary *b*	E1	E1	E2	E2	E2	E2	0
								Hydrocortisone
	<1	E1	E1	E1	E1	E1	E1	120·0
	1–2	E1	E1	E2	E2	E2	E3	80·0
	2–3	E1	E1	E1	E1	E1	E1	60·0
	3–4	E1	E1	E1	E2	E2	E2	60·0
7. Male aged 61	Preliminary *a*	E1	E1	E2	E2	E2	E2	0
	Preliminary *b*	E1	E1	E2	E2	E2	E3	0
								Prednisolone
	<1	E1	E1	E1	E1	E1	E1	30·0
	1–2	E1	E1	E2	E2	E2	E3	25·0
	2–3	E1	E1	E1	E1	E1	E1	20·0
	3–4	E1	E1	E2	E2	E3	E3	15·0
8. Female aged 40	Preliminary *a*	E1	E2	E2	E3	E3	E3	0
	Preliminary *b*	E1	E2	E2	E3	E3	E3	0
								Prednisone
	<1	E1	E1	E1	E1	E1	E1	25·0
	1–2	E1	E2	E2	E3	E3	E3	15·0
	3–4	E1	E2	E2	E3	E3	E3	15·0
9. Female aged 46	Preliminary *a*	E1	E1	E2	E3	E3	E3	0
	Preliminary *b*	E1	E1	E2	E3	E3	E3	0
								Prednisone
	<1	E1	E1	E3	E3	E3	E3	30·0
10. Male aged 52	Preliminary *a*	E1	E1	E3	E3	E3	E3	0
	Preliminary *b*	E1	E1	E2	E3	E3	E3	0
								Prednisone
	<1	E1	E2	E3	E3	E3	E3	15·0

SUMMARY

The skin response to ultraviolet light (U.V.L.) was studied in ten subjects with rheumatoid arthritis. The intensity and duration of the skin reaction was not modified by the systemic administration of corticosteroid preparations.

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We wish to thank Dr. H. S. Barber for permission to use patients under his care, and Mr. Butler, M.C.S.P., for his technical help.

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GUY'S HOSPITAL MEDICAL SCHOOL

DIPLOMA IN PHYSICAL MEDICINE—PART I

The next course for this Diploma will commence on Tuesday, January 6, 1959. Full particulars from: Physics Department, Guy's Hospital Medical School, London, S.E.1.

ORIGINAL PAPER

CHRONIC LEAD POISONING: MYOPATHY OR NEURITIS?

By DAVID PREISKEL

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INDUSTRIAL lead poisoning is very rare in Britain (Turner, 1955), and when it does occur it may be found in association with processes in which the presence of lead would not readily be suspected. It may involve workers in brass (alloy of copper–zinc) or in bronze and gunmetal (copper–tin), and Lauer (1955), who investigated 219 men in a brass foundry, found that 37 of them had had one or more acute episodes of lead intoxication. Their products were made of copper (75–85%), lead (5–15%), tin (5–10%), and zinc (1·5–5%). In the case described below the man had been polishing gunmetal castings made of copper (85%), tin (5%), zinc (5%), and lead (5%).

CASE REPORT

The patient, a man aged 60, had been a metal polisher for more than 16 years; in the past 3½ years he had polished gunmetal, and previously aluminium and stainless steel. He had been provided with a pint of milk a day at work, and an extractor unit was fitted to his bench.

On November 4, 1957, he was seen at the hospital, complaining that for three weeks he had had weakness of the wrists and was unable to hold things firmly. He was found to have some weakness of the wrist extensors and paralysis of both long extensors of the thumbs, more marked on the left side (Plate VIII). There were no sensory changes, and all tendon reflexes were present. His complexion could be described as saturnine, but no blue line was detected on the gums (edentulous).

On November 7 his haemoglobin level was 62% (Haldane). Red blood cells were normocytic with mild hypochromicity and with obvious polychromasia and punctate basophilia. A fasting specimen of urine showed increased coproporphyrins.

He took a chelating agent by mouth for three weeks (penicillamine, 0·3 g. thrice daily) and steps were taken to replace the grossly defective extractor unit on his work-bench. On January 2, 1958, his haemoglobin level reached 84% and no punctate basophilia could be detected. On February 6 the haemoglobin level was 90%. On February 20 he claimed that he felt much better and that his wrists were stronger; he looked much healthier, but no obvious change was found on examination of his wrist extensors.

ELECTRODIAGNOSTIC INVESTIGATION

26.11.57. Stimulation of the radial nerve on the left side produced responses in brachioradialis, abductor pollicis longus, extensor carpi ulnaris, extensor digitorum communis, and extensor carpi radialis. Direct stimulation of brachioradialis, extensor carpi radialis, and extensor carpi ulnaris did not reveal any abnormality. Extensor digitorum communis showed mild signs of denervation. However, electromyographic

exploration of extensor digitorum communis, extensor carpi ulnaris, and abductor pollicis longus revealed fibrillation at rest, while on volition a reduced motor-unit activity pattern was noted. The conduction velocity along the terminal portion of the radial nerve to the intact motor units appeared to be normal.

27.5.58. Stimulation of the left radial nerve with a current of 9·5 mA produced a response in abductor pollicis longus, brachioradialis, extensor digitorum communis, and extensor carpi ulnaris. The extensor pollicis longus did not respond. Intensity duration curves on the left extensor digitorum communis did not indicate denervation. On needle exploration there was no spontaneous activity, and on volition a complete pattern was produced composed of highly polyphasic potentials. A considerable degree of recovery seems to have occurred.

DISCUSSION

The classical work of Tanquerel des Planches (1839) on lead poisoning has influenced clinical thinking for nearly a century, but did not settle the mode, or rather the site, of action of lead. With the passage of time the only measure of agreement reached among various experts was that the problem was complex, and with the advent of modern laboratory methods it seems to have become even more complicated. More than thirty years ago a standard British textbook (Poulton, Symonds, and Barber, 1925) stated that "the paralytic symptoms are mainly due to a multiple neuritis". It was assumed that lead paralysis was probably due to a selective incidence of the poison upon the motor cells of the spinal cord. This seems to be in line with current French views that reaction of denervation is invariably present, and has been expressed very simply by Simonin (1950), who says: "Les paralysies saturnines sont des paralysies motrices pures, par atteinte nerveuse." In the U.S.A. Wechsler (1952) notes that "the central system is often involved, especially the anterior horn cells of the cord"; and in Germany Dennig (1952) adopts a somewhat similar view. Leading British neurologists appear to favour a dual concept; and Brain (1955), for example, assumes the existence of a lead "neuritis" which usually affects the extensor muscles of the wrist and fingers, and of a more diffuse type which attacks the central nervous system. Of the former he says that it is "better described as lead myopathy", and mentions that experiments suggest that lead has no direct action on the peripheral nerves. "Chronic cerebral symptoms and local and general progressive muscular atrophy in lead poisoning are, therefore, probably due to degeneration of the ganglion cells of the cerebral cortex and of the anterior horns of the spinal cord respectively." Another distinction is that "fibrillation, which is absent from the muscles paralysed in lead 'neuritis', is present in those degenerating through the action of lead on the anterior horn cells of the spinal cord". Stewart and Drought (1952) state that "lead palsy is the result of a peculiar form of lower motor neurone degeneration beginning in the muscle fibres and spreading upwards into the motor axons". Turner (1955)

writes that "lead neuritis is unlike any other type of peripheral neuritis in that it is purely motor (usually involving only limited groups of muscles), is not restricted to individual peripheral nerves or roots, and may involve either proximal or distal parts of the limbs". He is impressed by the fact that the muscles involved are those which the patient uses most constantly; in those carrying weights, for example, shoulder-girdle muscles may be paralysed. "These observations suggest that the effect of lead may be primarily on the muscles rather than on the peripheral nerves or nerve roots, and experimental evidence favours this view." He suggests that this is more likely to be caused by disturbance of resynthesis of phosphocreatine in the muscles (possibly by the action of lead in the intracellular enzyme system) than by neuropathy.

It is rewarding to examine some of the work that has been carried out on lead poisoning, and in particular that of Aub *et al.* (1925). Using mainly cats for their experiments, they were able to confirm the high efficiency of the liver in removing lead from the gastro-intestinal tract by means of the portal blood stream. They found that the concentration of lead in the skeleton progressively increased, while other tissues did not appear to retain the lead they received. By bathing isolated muscle with Ringer's solution containing 0·05 mg. of lead (as $PbCl_2$) per ml. it became clear that "leaded" muscle fatigued more readily and completely and recovered with much greater difficulty than control muscles. But the conductivity of the "leaded" nerve was exactly the same as that of the control, even after exposure to a concentration of lead as high as 0·46 mg. per ml.—the largest quantity soluble in Ringer's solution of *p*H 6·5. They were impressed by the clinical observation that the muscles paralysed are those which are most used, and were able to confirm this in their cats. Their post-mortem examination of human tissue revealed no lead in skeletal muscles, peripheral nerves, or cord, and tests carried out by refined technique showed that only minute quantities of lead were present in the blood at any given time. They came to the conclusion that "a satisfactory explanation, therefore, cannot be derived from pathological examinations alone" and made a plea for "dynamic methods of investigation". Adams and Weatherall (1954) investigated the affinity of various tissues for lead and found that it was highest in liver and bone marrow and low in muscle.

Experimentally, therefore, it has been shown that lead can exert a direct influence on muscle, but it does not follow that conditions in the human body have been exactly reproduced in the laboratory. No one knew this better than the skilled investigators who sighed for dynamic methods of research, but in their day, alas! they could not call upon that modern Oracle of Delphi the electromyograph, to speak from the depths (of living muscle) and give its verdict. The observation that fatigued muscle is more likely to become paralysed

has been fully attested by many observers and often quoted as evidence in favour of a lead myopathy. However, now that we know what happens to fatigued muscles in the invasive stage of acute anterior poliomyelitis this argument is not quite so impressive. The work of Sharrard (1953) has demonstrated the correlation between muscles paralysed in poliomyelitis and destruction of nerve cells found in the cord, where vertical columns of cells "represent peripheral musculature in muscle groups"; some of these columns are short, and therefore more liable to be put out of action, and others are long. It is therefore interesting to speculate on the observation that lead neuritis involves limited groups of muscles and is purely motor.

SUMMARY

A description is given of a case of chronic lead poisoning with paralysis of the extensor muscles of the wrists and fingers; clinically, it falls within the definition of so-called lead myopathy (lead "neuritis"). Electromyographic studies revealed no myopathic pattern. Were the muscles the primary site of the lead intoxication, with subsequent spread into the motor axons, the myopathy would be expected to mask the neuropathy; such was not the case. The findings were those of a motor polyneuritis. These findings, if generally confirmed, would give added support to those who maintain that lead causes paralysis by its action on nerve cells, and not on muscle.

ACKNOWLEDGMENTS

I am greatly indebted to Dr. P. Bauwens for carrying out the electrodiagnosis in his department, and for his helpful comments and encouragement. My thanks are also due to Dr. Victor Feldman for referring the case to me, to the pathological laboratory and physiotherapy staffs of the Hornsey Central Hospital for their co-operation, and to Miss E. M. Mason, the Group photographer.

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PLATE VIII

FIG. 1.—Maximal extension of left thumb.

FIG. 2.—Spreading fingers: no obvious wasting.

FIG. 3.—Escape of brachioradialis.

D.P. facing page 296]

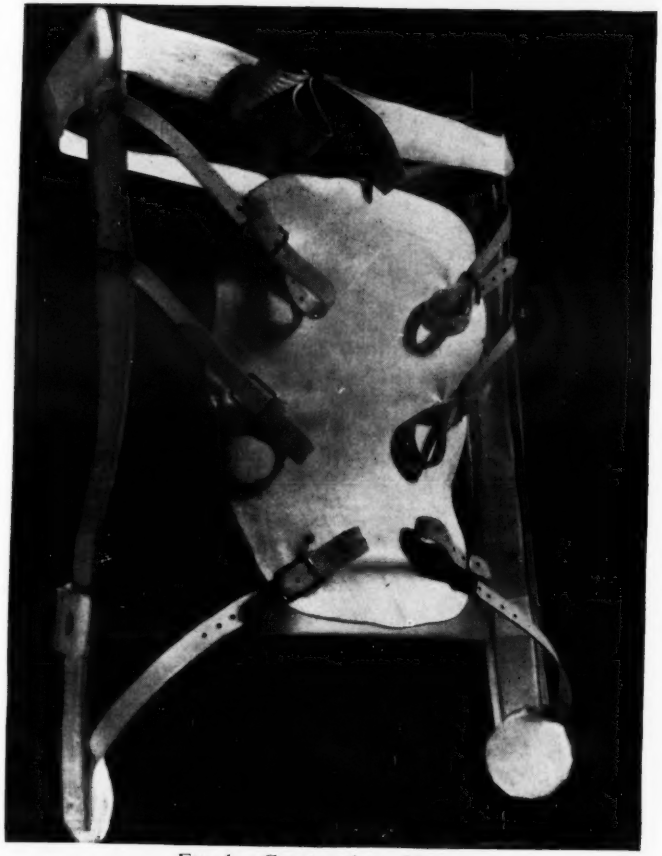

FIG. 1.—Construction of brace.

FIG. 2.—Maintenance of normal curve when patient tries to slump forward.

ORIGINAL PAPER

EXPERIENCES WITH AN EXPERIMENTAL SPINAL BRACE

By J. L. MILLIGAN

From the Royal Air Force Medical Rehabilitation Unit, Collaton Cross

THE work of Norton and Brown (1957) showed experimentally the ineffectiveness of many types of brace in immobilizing the lumbar spine. They designed an experimental brace which was more effective than most in producing the immobility desired under given test conditions, but noted, as a result of their work, that no form of support gave complete immobility of the lumbar spine.

The appliance is described here, and is recommended as a result of clinical trials undertaken at the Royal Air Force Medical Rehabilitation Unit, Collaton Cross. It has been found most effective in the conservative treatment of prolapsed intervertebral disks, in spinal anomalies, and after spinal fusion.

The brace consists of two horizontal bars, one at or just below the level of the posterior superior iliac spines and the other just below the level of the angle of the scapula. The bars are connected by lateral members in the mid-axillary line and extend downwards to the level of the greater trochanter of the femur, over which site they terminate in a padded disk. The brace is secured by a chest strap to the upper bar and by three straps from the lateral members to a central abdominal pad, the lowest of which is attached to the padded disk. The horizontal bars are made of polythene strips $1\frac{1}{8}$ inches wide and 5/32 inch thick, with duraluminum 17 SWG and 1 inch wide riveted to the strip and capable of being moulded to the spinal configuration. The duraluminum overlaps the polythene to prevent the latter bowing when the strip is moulded. The lateral bars are also of duraluminum $\frac{3}{4} \times \frac{1}{8}$ inch riveted to the ends of the horizontal strips. The whole is covered with leather (see Plate IX, Fig. 1). The abdominal pad, which is a large one extending from just below the xiphisternum to just above the pubis, is made of leather and chamois and is semi-rigid.

With this basic framework the following difficulties were encountered. The lower horizontal bar required a slight forward tilt at the upper edge to follow the contour of the line of the sacrum, and the bar had to be accurately shaped to fit snugly over the back of the sacrum and the part of the iliac crest which it crosses. The same careful fit to contour was necessary with the thoracic band. The side-bars had to be set strictly in the mid-axillary line and the lower end-pads accurately located over the greater trochanters. In this way difficulty is avoided when the patient sits down, the pad swivels slightly and

FIG. 2.—Maintenance of normal curve when patient tries to slump forward.

FIG. 1.—Construction of brace.

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allows the lowest strap to fall comfortably into the fold of the groin. The straps must be firmly secured to anchor the brace.

If care is taken over these points most patients can be comfortably fitted and any discomfort felt in the standing or sitting position is likely to be due to a bad fit. All the patients have been impressed by the comfort of this brace in normal wear and by the fact that it may be slept in with equal comfort. Because it is light and easily removed it allows washing or the taking of showerbaths.

From the treatment point of view, the brace has the added advantage that it does not replace the normal work of the spinal extensor muscles. This can be verified if it is worn next to the skin, when an excellent contraction of the spinal extensors can be seen. In addition the brace is a very potent reminder of poor posture, for in slump sitting or any form of spinal flexion, pressure over the posterior superior spine will cause much discomfort, resulting in the active maintenance of the normal lumbar lordosis. This is shown in Plate IX, Fig. 2, where the patient is trying to slump forward and the maintenance of the normal curve can be seen.

The braces used in the trials have been made in the unit, and could as easily be made by any local appliance manufacturer.

ACKNOWLEDGMENT

My thanks are due to the Director-General, Royal Air Force Medical Services, for permission to publish this work.

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CORRECTION

AUGUST NUMBER

In the paper on "Some Results of Rehabilitation of Injured Miners", by A. Zinovieff, page 258, line 1, it is stated that "all but 0·6% (or about one in every 200) are found a job in the pits". This refers to the discharge category, whereas the figure for the resettlement category—namely, 2·3% (or two in every 100)—should have been given.

REVISION SERIES

SYSTEMIC MANIFESTATIONS OF RHEUMATOID ARTHRITIS AND THE PARARHEUMATIC DISEASES*

III—LUPUS ERYTHEMATOSUS

By A. C. BOYLE

From the Department of Physical Medicine and Rheumatism, Middlesex Hospital

THE clinical manifestations of systemic lupus erythematosus (S.L.E.) are so protean that it is difficult to give an adequate description of the disease. It is a condition of unknown aetiology characterized by some or all of the following features: a constitutional illness often accompanied by fever and adenopathy; myalgia, arthralgia, or arthritis; erythematous skin lesions; suppression of blood-forming elements with leucopenia and anaemia, and widespread involvement of the viscera, particularly the kidneys, heart, and lungs. The disease has a curious predilection for the female sex, only about 10% of cases occurring in men, and, although it has been reported at almost any age, the peak incidence falls between 30 and 50 years.

Because of its varying manifestations it is difficult to form a concise picture of the frequency of its different clinical features; thus to the dermatologist it presents as a disease of the skin, with involvement of the viscera and sometimes the joints; to the general physician it presents as a general illness; while to the physician in physical medicine it presents as a disease of joints with involvement of the viscera and sometimes the skin. At one time it was considered to be an acute disease running a rapidly fatal course, but our whole concept of the disease has been altered by the discovery by Hargraves, Richmond, and Morton in 1948 of the L.E. cell phenomenon. This test, which appears to be nearly specific for the disease, has enabled us to detect a hitherto unsuspected group of cases which appear to be low-grade and which have a relatively good prognosis.

THE L.E. CELL TEST

The L.E. cell phenomenon is an entirely empirical test, depending upon the presence of an unknown substance in the plasma of patients with S.L.E. Under suitable conditions this substance induces a curious yet distinctive change in some leucocytes, and it is the recognition of the altered leucocyte which constitutes a positive L.E. cell test.

The plasma factor has been shown to be a component of the gamma-globulin fraction of the plasma proteins, and it appears to be quite distinct from the proteins which give rise to other serological anomalies, such as false

* Papers I and II appeared in the August number.

positive Wassermann reactions and positive cephalin and other flocculation tests. It is relatively heat-stable, being destroyed at 65° C., and is inactivated by a temperature of 4° C., although its activity may persist for several months even if the plasma is stored in a frozen state.

There are a number of methods for performing the L.E. cell test, but in all of them the basic principles are the same. Essentially, the test involves bringing the plasma or serum from the patient into contact with leucocytes, and after a suitable interval examining a concentrated film for the presence of L.E. cells.

The typical L.E. cell consists of a neutrophilic granulocyte distended by a large, homogeneous, globular inclusion body which compresses the segments of the nucleus against the cell membrane, leaving only a thin rim of cytoplasm (Conley, 1956). It is important to distinguish the true L.E. cell from Tart cells and other cells which may, to some extent, resemble the L.E. cell. In particular, it should be noted that the L.E. cell is larger than other leucocytes, that the inclusion body is smooth, structureless, and absolutely homogeneous, and that it stains slightly redder than the nuclei of the leucocytes. In addition, it is usual in a positive L.E. cell test to see rosettes and extracellular masses of depolymerized deoxyribose nucleic acid.

In some cases where a strong clinical suspicion of S.L.E. is held, yet the L.E. cell test is repeatedly negative, it may be possible to obtain a positive test by giving the patient 30 to 40 units of ACTH for two to three days and then repeating the test. Because of this, and because it has been suggested by Slocumb (1953) that reversion from rheumatoid arthritis to S.L.E. may occur following withdrawal of steroid therapy, we have recently attempted to produce the L.E. cell phenomenon in patients with rheumatoid arthritis on long-term steroid therapy by withdrawing treatment and searching for L.E. cells. In a small series so far we have not been able to produce a positive L.E. cell test by this method.

This brings us to the difficult question of the specificity of the L.E. cell test. Although occasional reports have appeared of positive tests in such conditions as penicillin sensitivity, glomerulonephritis, and liver disease, the real difficulty arises where a positive test occurs in what appears clinically to be an uncomplicated rheumatoid arthritis. I believe the L.E. cell test is specific for S.L.E., and, if a positive test occurs in rheumatoid arthritis, that either a careful search will reveal evidence of systemic disease to be present, or that the positive test heralds the development of systemic involvement in the future.

SKIN LESIONS

The skin is the classical site for the presentation of S.L.E., although in many cases skin lesions are absent throughout the entire course of the disease.

The typical butterfly rash on the face has been reported in less than 50% of patients in most large series of cases; sometimes bullous or ulcerating discoid lesions are seen in this area. Next in frequency appears to be the curious purplish erythema seen around the nail beds, and this seems to be particularly common in those patients with rheumatoid-type involvement of the finger-joints. Petechial haemorrhages and telangiectasia, mucocutaneous ulceration around the mouth with silvering of the lips, erythema nodosum, and purpura may be seen. Gravitational ulcers in the legs have been reported as a feature of the disease by Allison and Bettley (1957), though these are known to occur with undue frequency in rheumatoid arthritis. Alopecia is also described as a feature of S.L.E., although I have not seen an example of it among my own cases.

The relationship of chronic discoid lupus to S.L.E. has been a matter for debate, though most dermatologists seem now to be of the opinion that at any stage in chronic discoid lupus dissemination may occur, with eventual systemic manifestations of more widespread disease, often supported by the finding of a positive L.E. cell test.

The incidence of nodules of the rheumatoid type in S.L.E. is another matter which is open to debate. It is often said that they tend to be absent or minimal in S.L.E., whereas they occur in something like 25% of cases of rheumatoid arthritis. This is not my experience, for I believe that nodules which are microscopically indistinguishable from the nodules of rheumatoid arthritis are a common finding in S.L.E. In particular, the nodules appear often to be of the "necrobiotic" type, tending to break down and ulcerate from time to time, yet nevertheless healing quite quickly if protected from pressure.

In summary, it can be said that the butterfly rash and nail-bed erythema are the two most common skin lesions in S.L.E. Any type of skin lesion, however, may be seen, and it is a curious fact that the more disseminated the S.L.E. the more atypical do the skin lesions become.

ARTHRALGIA AND ARTHRITIS

Pain in muscles or joints occurs in over 70% of patients with S.L.E. Roughly speaking, these symptoms fall into three groups: (1) In the more acute cases of S.L.E. transient muscle and joint pain, often without objective signs, may appear to be no more than one manifestation of a severe constitutional illness. (2) Episodic arthralgia, diffuse muscle aching, or recurrent effusions into joints may herald the onset of a less acute type of S.L.E., and such symptoms may precede evidence of involvement of other systems by months or even years. (3) Established rheumatoid-type joints may be present for many years before evidence suggestive of S.L.E. becomes apparent. In such cases the course is usually chronic and the prognosis relatively benign. With this third type the

pattern of joint involvement is usually indistinguishable from true rheumatoid arthritis, and, although Keil (1940) suggests that ulnar deviation of the fingers is less common in S.L.E. than in rheumatoid arthritis, this is not in accordance with my own experience. There are, however, two features which should arouse the suspicion of S.L.E. with otherwise typical rheumatoid-type joints; these are: (1) subjective symptoms of pain and stiffness out of all proportion to the objective physical signs in the affected joints; (2) the radiological appearance of profound osteoporosis and extensive cartilage loss with curiously minimal or absent erosive changes.

PULMONARY LESIONS

Although the classical pulmonary lesion is pleural effusion often associated with other serous effusions, more attention has been focused recently on parenchymal lung involvement. Radiologically, the appearance is that of a diffuse fibrosis, most marked at the lung bases. Sometimes these changes are severe and widespread, yet, in contrast to the changes in the hands, often give rise to surprisingly little in the way of either symptoms or physical signs.

In 1944 Hamman and Rich described a condition characterized by diffuse fibrosis of the lung fields, now known as the Hamman–Rich syndrome, and it is perhaps significant that L.E. cells and other features of a collagen-like disease may have been demonstrated in a proportion of cases.

CARDIOVASCULAR LESIONS

Clinical evidence of involvement of the cardiovascular system is discernible in more than 50% of patients suffering from S.L.E. (Talbott and Ferrandis, 1956). In the heart recurrent fibrinous pericarditis, or an endocarditis of the Libman–Sacks type (Libman and Sacks, 1924), is well known, and may often be diagnosed during the course of the disease. Although a diffuse angiitis is an essential component of the pathology of S.L.E., gross vascular lesions are not commonly diagnosed during life, but occasionally there may be evidence of arterial obstruction followed by gangrene of the extremities. Raynaud's phenomenon does not occur so frequently in S.L.E. as in certain other collagen diseases, particularly scleroderma.

RENAL LESIONS

Although involvement of the kidneys has been reported as occurring in 100% of cases of S.L.E. (Montgomery and McCreight, 1949), it may be difficult to demonstrate during life in some cases, short of such a procedure as renal biopsy. The characteristic microscopic lesion is the wire-loop seen in the glomerulus, and is due to hyaline thickening of the basement membrane of the glomerular capillaries. Perhaps the most important feature of renal involvement

is that if demonstrated it denotes a bad prognosis. Unfortunately, it does not appear to be prevented or reversed by steroid treatment, but, on the other hand, there is no evidence that treatment with steroids in S.L.E. may give rise to a hypertensive state in the presence of renal involvement, as is sometimes the case in polyarteritis.

GASTRO-INTESTINAL LESIONS

These are usually clinically indefinite, giving rise to nausea or vomiting, anorexia, or vague abdominal pain, but occasionally a vascular episode may lead to more acute symptoms. Ascites may be part of a polyserositis.

NEUROLOGICAL LESIONS

Neurological lesions tend to be protean in character, since their basis is a diffuse angiitis. Epilepsy, hemiplegia, and peripheral neuropathy have all been described. In two of my patients confusion and intellectual deterioration were a marked feature and were presumably due to diffuse cerebral angiitis.

SUMMARY

In summary it can be said that S.L.E. is the commonest of the para-rheumatic diseases presenting in departments of physical medicine, by virtue of the frequency of myalgia, arthralgia, or arthritis as the predominant clinical feature; that S.L.E. should be suspected where subjective symptoms in the joints are out of proportion to objective signs of joint involvement, or where X rays show less destructive change than the clinical picture would suggest. Other common clinical manifestations suggesting S.L.E. in an apparent rheumatoid arthritis include persistent fever, albuminuria, systolic cardiac murmurs, or radiological signs of lung involvement.

Finally, although the L.E. cell test appears to be almost specific, the diagnosis of S.L.E. should rest on the clinical features of the disease and not on an isolated pathological finding; it is equally important to remember that a negative L.E. cell test by no means excludes the diagnosis.

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IV—POLYARTERITIS, SCLERODERMA, DERMATOMYOSITIS, AND THROMBOTIC THROMBOCYTOPENIC PURPURA

By S. MATTINGLY

POLYARTERITIS (PERIARTERITIS NODOSA)

POLYARTERITIS is a disease in which there are widespread inflammatory changes in the walls of small arteries, arterioles, and sometimes capillaries and veins. It is not as rare as textbooks suggest, but the diagnosis is often missed, especially in the early stages, because the clinical manifestations vary widely. More men are affected than women (3 : 1), usually between the ages of 20 and 25, but cases have been reported in infancy and old age. The aetiology remains unknown, but experimental work (Rich and Gregory, 1943; Hopps and Wissler, 1946) suggests that hypersensitivity, in particular to drugs such as the sulphonamides, may be responsible for the vascular changes. It may be significant that many patients give a history of bronchial asthma.

Necrosis of the media of small arteries may be confined to one segment of the arterial wall or may involve the entire circumference, spreading along the vessel or into the intima and adventitia, and aneurysms may develop which sometimes rupture. Microscopical examination reveals necrosis of the media and oedema of the vessel wall in the early stages; this is followed by fibrinoid degeneration and infiltration by polymorphonuclear leucocytes and eosinophil granulocytes, and later by plasma cells, lymphocytes, and monocytes; this granulation tissue is eventually replaced by fibrous tissue. Vessels may become completely occluded by scar tissue and thrombi, though recanalization can occur.

During the acute stage the disease may mimic an acute infection. Fever is usually present and low-grade, and is associated with generalized weakness and loss of weight. Skin lesions are common and varied, and include erythema multiforme, urticaria, and purpura. Occlusion of a major limb vessel results in gangrene. Subcutaneous nodules, which may be aneurysms or thickened segments of artery, are not common, especially in the early stages of the disease, and inasmuch as diagnosis can be confirmed by biopsy of a nodule this is unfortunate. They occur in crops on trunk or limbs, and even on the tongue, are painless, and persist a short time only, unless haemorrhage follows rupture of an aneurysm; they vary from a few millimetres to a centimetre or more in diameter.

Pain is a frequent symptom and may be localized or diffuse. Muscle tenderness is often severe and may suggest the diagnosis of trichiniasis or dermatomyositis. Joints may be painful and tender, but pain tends to be fleeting and

joint swelling is unusual. The typical arterial changes may be seen in affected synovia and capsule. Pain and paraesthesiae may be due to a peripheral neuritis, which develops in roughly half the patients with polyarteritis; it is often asymmetrical and may fluctuate in severity. Involvement of cranial arteries may result in hemiplegia, cranial-nerve palsies, or meningitis, and occlusion of the central artery of the retina or that supplying the visual cortex results in blindness.

Involvement of the mesenteric vessels gives rise to gastro-intestinal symptoms, and abdominal pain, vomiting, diarrhoea, and melaena may lead to laparotomy for suspected intestinal obstruction, perforated peptic ulcer, or appendicitis. Infarction and perforation of any part of the gut can occur. Occasionally excision of an affected appendix or gall-bladder results in apparent cure of the disease.

The renal arteries are frequently affected, and the patient may develop pain in the loin, dysuria, or haematuria and die from renal failure. A rising blood pressure associated with signs of renal damage suggests a very poor prognosis. Hypertension may be renal in origin, though it has been reported in patients without obvious involvement of the kidneys. It can result in hypertensive retinopathy and left ventricular failure. Although the coronary arteries are affected, angina pectoris and coronary thrombosis are not common.

With the exception of asthma, which may be present for years before the onset of polyarteritis, respiratory symptoms are uncommon, although the lung vessels are frequently affected. Occasionally pleural effusions, pneumonia, or lung abscess develop as complications of pulmonary infarcts, and these carry a grave prognosis. Radiographs of the lungs are usually normal.

The disease is often fatal, although some patients have survived, apparently in good health, for many years, even when the diagnosis has been confirmed by biopsy. Death usually results from damage to brain, heart, or kidneys.

Steroid therapy offers the only hope of suppressing the disease, but results are often disappointing even when symptoms are relieved, and there is some evidence that it causes further renal damage (Rich, Berthrong, and Bennett, 1950).

SYSTEMIC SCLERODERMA

Systemic scleroderma occurs at all ages, but most commonly in middle-aged women. The cause of this disease is not known; there is little evidence that it is allergic in origin, and, whilst it has been reported following a number of acute infections, these have been very varied and included malaria, typhoid, measles, and tuberculosis. It has been suggested that scleroderma is primarily a disease of the autonomic nervous system, because Raynaud's syndrome is often present in these patients before skin changes occur, and degenerative changes have been

reported in the sympathetic ganglia as well as in peripheral nerves. The results of sympathectomy have not supported this hypothesis.

Microscopical examination of the affected skin reveals thinning of the epidermis, with flattening of the rete pegs. If inflammatory changes occur at first, they are soon replaced by avascular fibrous tissue in the dermis, and the more specialized structures such as sebaceous glands and hair follicles disappear. Calcium may be deposited in the deeper layers of the dermis. It has been suggested that these changes in the skin are secondary to widespread obliteration of blood vessels, which are sometimes necrotic and similar to those seen in polyarteritis.

Any part of the alimentary tract from oesophagus to sigmoid colon may be affected, with atrophy of specialized tissues such as muscle and their replacement by fibrous tissue; the oesophagus may become a rigid tube, and thinning of the intestinal wall can lead to unexpected perforation and death from peritonitis.

The lungs are often affected, and fibrosis may be associated with the formation of cysts, possibly the result of bronchial obstruction and chronic infection. The pleura and pericardium may be thickened, and effusions be present in pericardial and pleural sacs. The myocardium often shows severe scarring apparently unrelated to occlusion of coronary arteries. The kidney is frequently the site of widespread vascular changes, and may be scarred and shrunken like an arteriosclerotic kidney; sometimes calcium is deposited.

It will be evident that the clinical manifestations of scleroderma are varied and not confined to the skin. The onset of symptoms is often insidious as in the other collagen diseases, and malaise, arthralgia, and Raynaud's syndrome may precede obvious skin changes by months or even years. A diagnosis of early rheumatoid arthritis is often made at this stage of the disease. The skin of the face, neck, or hands is usually affected first. A diffuse non-pitting oedema is soon followed by fibrosis; the skin hardens and cannot easily be separated from the subcutaneous tissues, and the joints, especially of the hands, become fixed in flexion. The skin of the face becomes firmly adherent to the underlying bony structure, skin folds are obliterated and movements of the jaw restricted. The face becomes like a mask or skull. If the disease persists for a year or more subcutaneous calcification develops, especially at the fingertips or in areas subject to pressure, and the overlying skin ulcerates and fails to heal.

Muscle weakness may be extreme owing to replacement of muscle by fibrous tissue. Dyspnoea may be due to lung fibrosis or myocardial fibrosis. Involvement of the gastro-intestinal tract results in dyspepsia, possibly due to oesophageal regurgitation, and dysphagia may become so severe that the patient is able to swallow only liquids. Inadequate intake of food associated

with its impaired absorption from the atrophic intestine results in severe malnutrition. Death, which may occur after months or years, is due to heart failure, sometimes associated with hypertension, renal failure, or intercurrent infection.

Addison wrote a graphic description of the disease nearly 100 years ago. "At length the part originally affected becomes more or less hide-bound, and a similar change taking place around the more superficial fasciae and tendons, the latter become so tightened, fixed and rigid, as to be no longer capable of performing their proper functions, and to such an extent, that the whole of a limb, but especially the fingers, may be permanently contracted, bent, and rendered almost as hard and immovable as a piece of wood; thereby impeding progression, distorting the gait, and making the patient a poor miserable cripple for the remainder of his life" (Addison, 1868).

DERMATOMYOSITIS

Dermatomyositis may be an acute or chronic disease. It affects both sexes, usually between the ages of 30 and 50, although children not infrequently develop acute dermatomyositis. The aetiology, like that of the other collagen diseases, is as yet unknown, but in about 20% of cases dermatomyositis may be associated with a malignant tumour; this may be a carcinoma or sarcoma affecting any organ, or even Hodgkin's disease and myelomatosis. It has been claimed that successful treatment of the neoplasm may cure the dermatomyositis, but Talbott and Ferrandis (1956) consider the evidence unconvincing.

Myositis is the dominant feature of the disease. The muscle fibres disintegrate and the muscle becomes swollen with oedema and infiltrated with lymphocytes, plasma cells, and monocytes; vessels may be affected, but the necrotic lesions of polyarteritis are not seen. Damaged muscle is replaced by fibrous tissue. Skin changes are sometimes indistinguishable from those of scleroderma, and include calcinosis, and some physicians believe that dermatomyositis and scleroderma are variants of the same disease.

The onset is sometimes acute, but more often insidious, with low-grade fever, painful muscles, and even swollen joints, suggesting the diagnosis of rheumatic fever or rheumatoid arthritis. There may be patches of erythema over affected muscles or on the face, where they may simulate the characteristic butterfly rash of systemic lupus erythematosus; swelling of the eyelids is characteristic, but this periorbital oedema may persist for only a few days. Later the skin may become pigmented and atrophy and closely resemble scleroderma. Weakness of the skeletal muscles may be marked; the proximal limb muscles are commonly affected, are sometimes but not always tender, and

may become so weak that the patient is unable to walk or even to sit up in bed. If the patient survives long enough, fibrous contractures of joints develop. Fibrosis of the lungs and weakness of intercostal muscles may result in intercurrent chest infections or heart failure, while atrophy of the tongue and pharyngeal muscles makes speaking and eating difficult. Involvement of the gastro-intestinal tract, as in scleroderma, results in dysphagia and dyspepsia. Signs of a peripheral neuritis may be present, as well as mental confusion and delirium, and such features account for the use of the term "neurodermatomyositis".

Most patients die within a few years of the onset of dermatomyositis, and, in the acute stage of the disease, death can occur after two or three months, from intercurrent infection, heart failure, or renal damage. Remissions sometimes follow removal of a malignant tumour or steroid therapy, and some patients survive for many years suffering from extensive contractures and malnutrition.

THROMBOTIC THROMBOCYTOPENIC PURPURA

The last of these four so-called "collagen diseases" is thrombotic thrombocytopenic purpura, or T.T.P. It is very rare. It is thought to be a generalized vascular disease affecting the intima of all small blood vessels, especially at the junction of arterioles and capillaries (Talbott and Ferrandis, 1956). Allergy to drugs and micro-organisms has been suspected, but no cause is known.

An amorphous and finely granular substance, possibly derived from the cement between the endothelial cells, accumulates beneath the endothelium, protrudes into the lumen of the vessel, and becomes covered with adherent platelets. Occlusion of vessels results in multiple thrombi, and thinning of the vessel wall leads to aneurysms. The inflammatory changes of polyarteritis have not been seen. Examination of the blood reveals a haemolytic anaemia and thrombocytopenia.

The symptoms may be very varied, with fever, weakness, loss of weight, and muscle and joint pain. Rheumatic fever or rheumatoid arthritis may be suspected at first, and some patients with proven systemic lupus erythematosus appear to develop T.T.P. However, severe haemorrhages from skin and mucous membranes, jaundice, hepato-splenomegaly, and fluctuating neurological signs in an acutely ill patient will suggest the diagnosis of thrombotic thrombocytopenic purpura. The differential diagnosis includes a number of blood diseases, such as idiopathic thrombocytopenic purpura, acquired haemolytic anaemia, and leukaemia, as well as polyarteritis and subacute bacterial endocarditis. Death usually occurs in a few months and sometimes within weeks of the onset of symptoms.

CONCLUSIONS

The rarer collagen diseases should be considered in the differential diagnosis when patients present with obscure complaints of joint or muscle pain, especially when these are associated with skin lesions, fever, hypertension, and renal disease.

We as yet know nothing of their aetiology or pathology, although it seems that when inflammatory changes occur they are probably secondary to necrosis of collagen. Distinct clinical patterns exist, and whilst they may present with similar symptoms and share some rather non-specific pathological changes these do not necessarily indicate a common aetiology or pathogenesis.

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REVIEWS OF BOOKS

SUSPENSION THERAPY IN REHABILITATION. By M. Hollis and M. H. Roper. *Pp. 220. 25s. London: Baillière, Tindall and Cox. 1958.*

This book is in two parts. The first part covers the history of sling suspension exercises, the apparatus needed, the physical principles involved, and the many techniques used. All this is written in a clear and attractive style, and it is profusely illustrated with excellent diagrams. This half of the book can be recommended to anyone interested in the physical aspects of rehabilitation.

The second part is devoted to the application of the principles to clinical conditions, with chapters on orthopaedic, rheumatic, geriatric, and neurological conditions, spinal paraplegia, and amputations. Unfortunately, most of these chapters do not help as much as they might. Some of them are so filled with details of general management that suspension exercises are scarcely mentioned. "Rehabilitation after Amputation" is quite outstanding; it gives an excellent account of the general problems involved, and also gives details of the suspension and pulley exercises and how they fit into the rehabilitation programme. "Suspension Therapy and the Geriatric Patient" is also good and contains many practical ideas. When this book goes into a second edition, as it should, firm editing of the second part will be needed. Despite this criticism the volume is strongly recommended, and it is hoped that more physiotherapists will be encouraged to write books of this calibre.

D. A. BREWERTON

THE CLINICAL PHYSIOLOGY OF PHYSICAL FITNESS AND REHABILITATION. By Ernest Jokl, M.D. *Pp. 194. 63s. Oxford: Blackwell Scientific Publications. 1958.*

In his introduction Dr. Jokl points to the importance of exercise in rehabilitation, and complains that it has been founded on empirical data rather than sound physiological and clinical theory. Unfortunately his book does little to correct this, for it is based mainly on personal views and observations rather than on the wide research that has been done on this subject in recent years.

Dr. Jokl is director of a rehabilitation centre in Kentucky, and obviously has wide experience of medicine, physiology, gymnastics, athletics, and music, and it is this that makes his book interesting. He has a refreshing insistence on movement and skill, as opposed to exercise for individual muscles and joints. He stresses the importance of sensation and the sense of movement, and never fails to illustrate the complexity of the physiological adaptations that followed repeated exercise.

Anyone particularly interested in rehabilitation and exercise therapy would be sure to get a number of new ideas by reading this book, but it cannot be recommended as a reliable and comprehensive guide to the subject.

D. A. BREWERTON

ABSTRACTS OF THE LITERATURE

The Effect of Ageing on Plantar Flexor and Superficial Abdominal Reflexes in Man. A Clinical and Electromyographic Study. J. W. MAGLADERY, R. D. TEASDALL, and A. H. NORRIS. *J. Geront.*, 1958, **13**, 282.

It is well recognized that motor responses in old age are depressed, but the physiological mechanism of this has, so far, escaped definition. Latency in simple myotatic reflexes is somewhat increased in older animals, but this slight degree is not demonstrable in man. However, two cutaneous reflexes which are influenced from the higher centres may be susceptible to a comparative study.

The present investigation showed that latencies in the normal plantar flexor response and superficial abdominal reflexes were increased in older age groups. The reason for this is discussed, and the authors believe that it is due, not to alteration in the basic reflex mechanisms themselves, but to decreased excitatory influence on them from higher levels. G. O. STOREY

Cervical Disk Lesions. G. L. ODOM, W. FINNEY, and B. WOODHALL. *J. Amer. med. Ass.*, 1958, **166**, 23.

The authors review 246 cases of cervical disk protrusion or foraminal spur operated on during a sixteen-year period at Duke Hospital, Durham, North Carolina. All of the cases had been investigated carefully, both clinically and radiologically, and these findings were compared with the operative findings. Myelography was shown to be reliable, but clinical examination and routine radiography were not at all reliable in placing the level of the lesion. [This article is well worth reading in the original.]

D. A. BREWERTON

Stress Fractures of the Tibia in Athletes, or "Shin Soreness". M. B. DEVAS. *J. Bone Jt Surg.*, 1958, **40B**, 227.

This paper stresses that "shin soreness" in athletes is caused by a fracture involving one cortex of the tibia and is not apparent on radiographs until up to three months after the onset. The symptoms start insidiously with aching in the shin after exercise, gradually increasing in severity. Symptoms settle with rest, only to recur after further athletic activity.

A series of 16 cases seen at the Middlesex Hospital Athletic Clinic are reviewed together with an analysis of 78 cases reported in the literature. The diagnosis, treatment, and mechanism of this particular type of stress fracture are discussed in detail. The importance of early institution of rest is emphasized. P. J. R. NICHOLS

Acroparaesthesia in Myxoedema. I. P. C. MURRAY and J. A. SIMPSON. *Lancet*, 1958, **1**, 1360.

In a group of 35 consecutive patients suffering from proven myxoedema 26 patients were found to have acroparaesthesia. Clinical and electromyographic studies showed this to be due to a median neuritis probably caused by accumulation of myxoedematous tissue under the flexor retinaculum at the wrist. Treatment with thyroxine usually relieved the symptoms.

S. MATTINGLY

Multiple Epiphysial Dysplasia. H. BARRIE, C. CARTER, and J. SUTCLIFFE. *Brit. med. J.*, 1958, **2**, 133.

Multiple epiphysial dysplasia is described as a genetically determined condition in which there is irregular and often retarded ossification in several of the epiphyses; disability may be severe, with recurrent attacks of joint pain and stiffness and eventually osteoarthritis. The authors review 15 cases occurring in three families, and point out that the diagnosis is frequently missed, although radiographs are diagnostic. Treatment is empirical, but the authors favour non-weight-bearing exercises, including swimming, especially in the early stages of the disease. S. MATTINGLY

Intra-articular Therapy in Rheumatoid Arthritis. G. N. CHANDLER, V. WRIGHT, and S. J. HARTFALL. *Lancet*, 1958, **2**, 659.

A "double-blind" crossover trial of the effects of intra-articular injection in the local treatment of rheumatoid arthritis was planned to assess the comparative value of hydrocortisone acetate and tertiary butyl acetate (T.B.A.), and an inert substance. The knees of 24 patients were injected. Both steroids relieved symptoms and improved function to a greater extent than did the placebo, but improvement was usually

greater and of longer duration with hydrocortisone T.B.A. than with hydrocortisone acetate. However this difference was not statistically significant.

[The results of the trial suggest that there is some advantage in using hydrocortisone T.B.A. for intra-articular injections in rheumatoid arthritis, but the authors do not clearly state their own conclusions, though it appears that they were to the contrary.]

S. MATTINGLY

Deleterious Effect of Intra-articular Hydrocortisone. G. N. CHANDLER and V. WRIGHT. *Lancet*, 1958, **2**, 661.

The authors suggest, from a radiological study of 18 patients with rheumatoid arthritis, that the injection of hydrocortisone into the knee-joint may, by relieving symptoms, encourage overuse of a damaged joint, and as a result the joint deteriorates more rapidly than it would if not injected.

S. MATTINGLY

Therapeutic Application of Ultrasound in Physical Medicine. J. F. LEHMANN and F. H. KRUSEN. *Amer. J. phys. Med.*, 1958, **37**, 173.

In this paper, read at the International Conference on Ultrasonics in Medicine, held at Los Angeles in September, 1957, the authors summarize the techniques of application of ultrasound and the dosages used. In discussing the indications for treatment they group the conditions into those in which ultrasound is of established value, of suggested value, and of questionable value. Ultrasound is considered to be of established value in periarthritis of the shoulder; of suggested value in rheumatoid spondylitis, painful neuroma, reflex dystrophy, Dupuytren's contracture, and for pain relief; and of questionable value in sciatica, neuritis, epicondylitis, tenosynovitis, arterial insufficiency, ulcers, and "a host of conditions".

The authors conclude that ultrasound is a useful form of physical therapy, and that it can be used safely and with biophysical knowledge of its effects, that the indications and contra-indications for its use are essentially those of other forms of deep heat, but that specific effects may well occur in some conditions. Objective clinical evidence is, however, most scanty compared with the amount that has been written on the subject.

D. C. ARNOTT

Triamcinolone. F. DUDLEY HART, J. R. GOLDING, and D. BURLEY. *Lancet*, 1958, **2**, 495.

Taking 4 mg. of triamcinolone to be equivalent to 5 mg. of prednisolone, the authors conclude from a short-term study that these steroids do not differ greatly in therapeutic action or side-effects. However, additional side-effects were noted with triamcinolone in three patients: the appearance of abdominal striae and post-prandial flushing.

S. MATTINGLY

Triamcinolone Arthritis. R. WELLS. *Lancet*, 1958, **2**, 498.

Three patients who had been treated for over six months with prednisolone for thrombocytopenic purpura and asthma developed arthritis when prednisolone was replaced by triamcinolone. The author suggests that arthritis may be a side-effect of triamcinolone therapy.

[In each case the dose of triamcinolone used was less than half that of prednisolone, and joint symptoms subsided when prednisolone was once more given in the original dosage. It seems likely that the "arthritis" was related to a reduction in steroid dosage rather than a change of drug.]

S. MATTINGLY

The Results of Partial and Total Excision of the Patella. H. L. DUTHIE and J. R. HUTCHINSON. *J. Bone Jt Surg.*, 1958, **40B**, 75.

Opinions differ on the effects of excision of the patella. In this paper from the Western Infirmary, Glasgow, are reviewed the results of partial and total excision of the patella for fractures and chondromalacia. The cases are all from one unit and have been followed up for one to ten years. The results are analysed in detail according to the operation, the reason for operation, and symptoms and findings at follow-up.

It is interesting that no satisfactory explanation for subjective weakness following total excision was found. After partial excision, however, weakness was related to radiological evidence of arthritis. The findings lead the authors to recommend total excision when patello-femoral arthritis is already present.

[The results in this series fall between the extremes of other published reports and thus form a useful basis for comparison.]

P. J. R. NICHOLS

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